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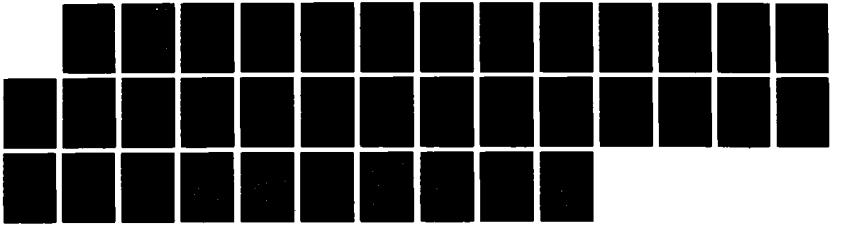

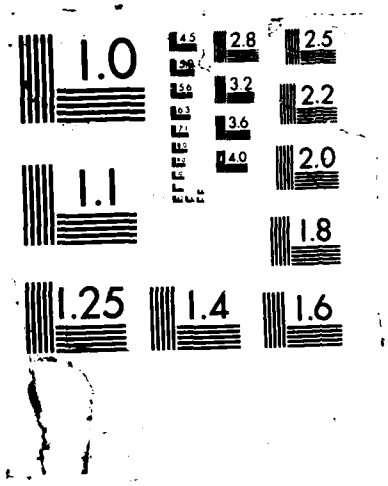
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RESPIRATORY AND CARDIOVASCULAR RESPONSES TO COLD STRESS FOLLOWING REPEATED COLD WATER IMMERSION

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The effects of cold acclimation (CA) on the cardiorespiratory responses to cold air and water stress tests (CST) were studied in seven males before and after a CA program of daily 90 min cold water (18°C) immersions repeated five times a week for five consecutive weeks. The CST consisted of a 90 min resting exposure to cold air (5°C, 30% rh) or water (18°C) during which rectal temperature (T_{re}), oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), minute ventilation ($\dot{V}_E$), heart rate (HR), cardiac output ($\dot{Q}$) and blood pressure (BP) were periodically measured. In cold air following CA, the $\dot{V}O_2$ at 10 minutes was lower ($p < 0.02$) post than pre CA, however, no differences were found in cold water. The $\dot{V}_E$ increased ($p < 0.01$) during CST as a function of $\dot{V}CO_2$. The CA did not affect the $\dot{V}_E$-$\dot{V}CO_2$ relationship or the pattern of breathing during CST in cold air or water. The CA had no effect on $\dot{Q}$ or $(a-\bar{v})O_2$ difference, which both increased ($p < 0.01$) during the first 45 min of CST then remained stable. BP increased significantly during the first cold water exposure, but not during the last cold water immersion. These data indicate that CA attenuated the

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onset of metabolic heat production during CST in air but did not alter its ultimate magnitude or the relationships between the cardiorespiratory variables and metabolic requirements. Also, the thermoregulatory adjustments associated with CA altered the control of blood pressure during acute cold stress.

ABSTRACT

The effects of cold acclimation (CA) on the cardiorespiratory responses to cold air and water stress tests (CST) were studied in seven males before and after a CA program of daily 90 min cold water (18° C) immersions repeated five times a week for five consecutive weeks. The CST consisted of a 90 min resting exposure to cold air (5° C, 30% rh) or water (18° C) during which rectal temperature (T_{re}), oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), minute ventilation ($\dot{V}_E$), heart rate (HR), cardiac output ($\dot{Q}$) and blood pressure (BP) were periodically measured. In cold air following CA, the $\dot{V}O_2$ at 10 minutes was lower ($p<0.02$) post than pre CA, however, no differences were found in cold water. The $\dot{V}_E$ increased ($p<0.01$) during CST as a function of $\dot{V}CO_2$. The CA did not affect the $\dot{V}_E$-$\dot{V}CO_2$ relationship or the pattern of breathing during CST in cold air or water. The CA had no effect on $\dot{Q}$ or $(a-\bar{v})\,O_2$ difference, which both increased ($p<0.01$) during the first 45 min of CST, then remained stable. BP increased significantly during the first cold water exposure, but not during the last cold water immersion. These data indicate that CA attenuated the onset of metabolic heat production during CST in air but did not alter its ultimate magnitude or the relationships between the cardiorespiratory variables and metabolic requirements. Also the thermoregulatory adjustments associated with CA altered the control of blood pressure during acute cold stress.

KEY WORDS: Hypothermia, body cooling, ventilatory responses, cold water, cold air

INTRODUCTION

The respiratory responses to cold stress have received relatively limited study and generally cover only single, acute, cold exposures. Generally, minute ventilation increases progressively in response to the increasing metabolic demands of the cold stress (1,2,3,4). Resting minute ventilation has been reported to nearly double during an exposure to 8° C air for two hours (4). The intensity of this ventilatory response is inversely related to the water temperature (5) and may play an important role in determining whether the victim inhales water. The control of the pattern of breathing during mild hypothermia has not been studied. Also, the effect of mild hypothermia on the chemoreceptor inputs to the control of breathing is not known. Nor has the respiratory response to cold stress before and after acclimation to cold been investigated.

The cardiovascular responses to cold stress vary depending upon the nature of the cold stress (air vs. water, whole body vs. local exposure, ambient temperature and duration of exposure) and the metabolic and neurophysiological status of the organism being stress. Generally, deep hypothermia (core temperature less than 30° C) causes a reduction of arterial blood pressure, heart rate, cardiac output, total peripheral resistance and oxygen consumption (6,7). However, in normothermic or moderately hypothermic conscious humans and unanesthetized animals, cold stress elicits quite different cardiovascular responses (1,2,8,9,10,11,12). Virtually all of these studies have demonstrated an increased mean arterial blood pressure via increases in either systolic blood pressure (8),

diastolic blood pressure (9) or both (1,2,10,11,12). However, the heart rate response to cold stress appears to be more variable and not apparently associated with the nature of the cold stress (i.e., air vs. water) (1,2,3,4,8,9,10,12,13,14,15,16,17). Several studies have measured an increased cardiac output in response to cold stress (1,3,8,12,13,17), generally attained via an increased stroke volume (3,8,12,13,17). The elevated stroke volume has been attributed to an enhancement of venous return via increased peripheral and cutaneous vasoconstriction as the body cools (12,17). However, calculated total peripheral resistance has been shown to increase (9,17) decrease (8,12) or remain constant (13) in response to cold stress.

All of the aforementioned studies of cardiovascular responses to cold stress were one-time acute exposures. Only two studies, Budd et al. (10) and Hong et al. (15) investigated the effect of acclimation on cardiovascular responses to cold stress. Both studies limited their observations to blood pressure and/or heart rate responses. Budd et al. observed that after cold acclimation the systolic blood pressure response to cold stress was attenuated, whereas the bradycardia was enhanced. Likewise, Hong et al. (15) observed enhanced bradycardia during winter diving in the Korean Ama compared to summer diving. No investigations have studied whether the cardiovascular responses to cold are altered during the process of acclimating to cold. One would predict that if the increased cardiac output is serving the increased oxygen demand of the metabolic response to cold stress, as the metabolic response changes with acclimation (18,19,20,21,22) likewise the cardiovascular responses should change.

In most previous studies, the respiratory and cardiovascular responses to cold stress were measured at a single point in time or derived from data aggregated over time. Consequently, the results do not accurately describe the changing nature of the physiological responses during the exposure to cold stress. Furthermore, in many studies the physiological responses to the cold stress are reported as a function of the ambient environment and not as a function of the organism's thermal status. Thus, little information is readily available regarding the respiratory and cardiovascular responses as a function of core temperature during whole-body cooling. This investigation examined, under controlled laboratory conditions, the effects of cold acclimation on human cardiorespiratory control during acute cold water and air exposure.

METHODS

Subjects and experimental design

The subjects were seven male Caucasians native to the continental United States. They were fully informed about the requirements and risks of participation. Descriptive characteristics of the subjects were (mean ± SE): age = 24±2 yr; body mass = 70±4 kg; body surface area = 1.98±0.07 m^2; body fat (hydrostatic weighing (23)) = 17.4±1.8%; mean skinfold thickness (14 sites) = 11.4±1.5 mm; maximum aerobic power (treadmill running (24)) = 45.3±1.6 $ml \cdot kg^{-1} \cdot min^{-1}$. The study was conducted in Natick, MA, during the late fall when seasonal effects of cold exposure were expected to be minimal. For the 9 months preceding the study the subjects had not participated in any

significant cold weather activities. Subjects abstained from food, beverages, except for water, and tobacco for at least 2 h before reporting for any tests. All experimental procedures were performed at the same time of day for a given subject throughout the study.

Subjects completed a standardized cold air stress test (CST) 2 days before and again 2 days after completion of a cold acclimation program. The CST consisted of a 30 min baseline period spent reclining on a nylon-mesh lounge chair in a comfortable environment (T_a = 24° C, relative humidity (rh) = 30%) while wrapped in blankets. After the baseline period the subject stood and entered the environmental chamber (T_a = 5° C, rh = 30%), and then reclined for 90 min, wearing only swim trunks. During the last 5 min of both periods, venous blood samples were obtained from an indwelling catheter previously placed in the antecubital vein for determination of plasma norepinephrine (NE) and epinephrine (E). Oxygen uptake ($\dot{V}O_2$, $l \cdot min^{-1}$, STPD), carbon dioxide production ($\dot{V}CO_2$, $l \cdot min^{-1}$, STPD), minute ventilation ($\dot{V}_E$, $l \cdot min^{-1}$, BTPS), cardiac output ($\dot{Q}$, $l \cdot min^{-1}$), heart rate (bpm) and blood pressure (mmHg) were determined once during the baseline period and periodically during the cold air exposure. Rectal (T_{re}) and skin (T_{sk}) temperatures were measured during the last 3 min of the baseline period and at 2 min intervals throughout the cold exposure.

The repeated cold water immersion program consisted of a daily, 90 min immersion in cold (18° C, stirred) water, repeated 5 times a wk for 5 consecutive wk. In general, the acclimation sessions were accomplished on 5

consecutive days each week (Monday-Friday) followed by a 2 day rest. However, on occasion an individual missed a scheduled immersion which was made-up on the weekend. Due to a midweek holiday an immersion was missed which could not be rescheduled. Thus, the subjects completed a total of 24 water immersions. The water immersions were performed at the same time of day as the CST in air was performed. For each session, the subject reclined quietly on a nylon-mesh lounge chair while immersed to the neck in the water. Physiological responses were measured during the first and last cold water immersion sessions according to the same protocol as the cold air exposure, with the exception that the baseline environmental conditions were slightly warmer (T_a = 27° C, rh = 65%). During all cold water immersions, T_{re} was continuously monitored. The cold water immersions were terminated after 90 min or if the T_{re} fell below 35° C after which the subject was dried and rewarmed.

Experimental procedures

Chest electrodes (CM 5 placement) were used to obtain electrocardiograms, which were radiotelemetered to an oscilloscope-cardiotachometer unit (Hewlett-Packard) for measurement of heart rate. Ventilatory parameters, $\dot{V}O_2$ and $\dot{V}CO_2$ were measured using an automated open-circuit spirometry system (Sensormedics Horizon MMC). Cardiac output was measured using a semi-automated system (Sensormedics Horizon MMC) employing the CO_2 rebreathing method (25). Blood pressure was measured by auscultation. A thermistor inserted 10 cm beyond the anal sphincter was used to measure T_{re}. Skin

temperature was measured at three sites using thermocouples taped to the skin (forearm, chest and calf); $\bar{T}_{sk}$ and $\bar{T}_b$ was calculated (26).

Statistical analyses

Multifactor, repeated measures analysis of variance (ANOVA) was used to determine if the factors "exposure" (baseline vs. cold exposure) or "acclimation" (pre vs. post) had significant effects. In the event that ANOVA revealed significant main effects or multifactor interactions, Tukey's critical difference was calculated and used to locate significant differences between means. Single factor regression analysis was performed to compare the changes in cardiorespiratory parameters with changes in metabolic rate and body temperature. Results are presented as the mean ± SE. The results obtained from the thermoregulatory data and vascular fluid responses have been presented and discussed in detail elsewhere (22,27).

RESULTS

Cold Air Exposures

The oxygen uptakes and carbon dioxide productions are presented as functions of duration of cold air exposure in Fig. 1. Within the first 10 min of cold exposure prior to the cold acclimation program, the subjects' metabolic rate increased ($p<0.01$) by approximately 93%. Postacclimation the oxygen uptake was significantly lower than the pre-acclimation oxygen uptake at 10 min. Carbon dioxide production was not effected by acclimation. As shown in Fig. 1, after cold acclimation the respiratory exchange ratio was significantly

($p<0.05$) elevated during the baseline and initial 40 min of cold air exposure compared to the preacclimation values.

In Fig. 2 the minute ventilation, tidal volume and respiratory frequency responses to cold air exposure as a function of time are presented. These variables were not effected by cold acclimation. The effect of cold acclimation on the relationship between minute ventilation and tidal volume during cold air exposure is presented in Fig. 3. Cold acclimation did not alter the pattern of breathing during cold air exposure. Although tidal volumes increased proportionally more than respiratory frequency, in no subject did tidal volume increase to reach a maximum value (>50% vital capacity).

Further analysis of the effect of cold stress on the control of breathing is presented in Table 1. Both pre and post acclimation the minute ventilation was highly correlated ($p<0.01$) to carbon dioxide production. Cold acclimation did not alter the relationship between minute ventilation and carbon dioxide production. Postacclimation, the minute ventilation was inversely related to mean body temperature ($p<0.05$) and mean weighted skin temperature ($p<0.01$). However, no relationship was observed before or after acclimation between minute ventilation and rectal temperature during cold air exposure.

The cardiac output, stroke volume and heart rate values as a function of time during cold air exposure are presented in Fig. 4. These variables were not effected by cold acclimation. The effects of cold exposure and acclimation on the control of cardiac output are listed in Table 2. Cardiac output was

strongly correlated ($p<0.01$) to oxygen uptake during cold air exposure pre and post acclimation. This relationship was unchanged by the cold acclimation program. Before or after cold acclimation no significant correlations were obtained between cardiac output and mean body temperature, rectal temperature or mean weighted skin temperature. The calculated $(a-\bar{V})\ O_2$ difference increased ($p<0.01$) from 5.2 ± 0.4 to 9.0 ± 0.7 ml% during the first cold air exposure and from 4.8 ± 0.3 to 9.6 ± 1.1 ml% during the postacclimation cold air test. Acclimation did not alter the magnitude of the $(a-\bar{V})\ O_2$ difference response to cold air stress.

Due to an oversight, blood pressure was not measured during the first cold air exposure. Following the acclimation program, mean arterial blood pressure did significantly ($p<0.01$) increase, but only after 45 min of cold exposure (94.4 ± 3.0 and 106.3 ± 2.7 mmHg, at baseline and 80 min exposure respectively). Total peripheral resistance (TPR) was 0.016 ± 0.002 and 0.012 ± 0.001 $mmHg \cdot ml^{-1} \cdot min^{-1}$ prior to and after 80 min of cold air exposure. The TPR tended ($p=0.06$) to be lower than baseline during cold air exposure.

Cold water exposures

The oxygen uptake, minute ventilation and cardiac output values as a function of time during the first and last (24th) cold water exposures are presented in Fig. 5. These variables were not altered by cold acclimation. Tidal volume and respiratory frequency increased ($p<0.01$) by 86 and 20% respectively preacclimation, and 61 and 36% respectively postacclimation. The relationship between minute ventilation and carbon dioxide during cold water

immersion (Table 1) demonstrated a high correlation ($p<0.01$) pre and post acclimation. Furthermore, cold acclimation resulted in a significant ($p<0.05$) increase in the slope of the relationship from 30.9 to 35.3 (l, BTPS/l, STPD). Inverse relationships between minute ventilation and mean body and rectal temperatures were observed pre and post acclimation.

The cardiac output increased during the first 45 min of the cold water immersions (Fig. 5). No differences ($p>0.05$) existed between the 1st and 24th water immersions. Prior to the last cold water immersion, the baseline cardiac output tended to be slightly higher ($p\approx0.1$) than the respective preacclimation value. Consequently, although the cardiac output response during the cold water exposure was unchanged pre vs. post acclimation, the percent change was diminished. The increased cardiac output *was almost solely* due to increased stroke volumes, since heart rates were not significantly increased. As previously described for the cold air exposures, a strong correlation was observed between cardiac output and oxygen uptake during the cold water immersions (Table 2). However, unlike the cold air exposures, significant inverse correlations were obtained between cardiac output and mean body and rectal temperatures during the 1st and 24th water immersions. Cold acclimation did not significantly alter these relationships. The calculated $(a-\bar{V})\ O_2$ difference were 5.0 ± 0.7 and 4.8 ± 0.3 ml% for the pre and post acclimation baseline periods. During cold water immersion, the $(a-\bar{V})\ O_2$ difference increased ($p<0.01$) to 9.0 ± 0.6 and 9.4 ± 0.9 ml% pre and post acclimation respectively.

The mean arterial blood pressure responses to cold water immersion as a function of time are presented in Fig. 6. During the 1st immersion mean arterial blood pressure significantly increased from baseline due to increases of both systolic and diastolic blood pressures. There was a significant acclimation effect ($p<0.01$) with lower values postacclimation. Both pre and post acclimation TPR did not systematically change during cold water immersion. Baseline TPR was 0.016 ± 0.003 and 0.012 ± 0.001 $mmHg \cdot ml^{-1} \cdot min^{-1}$ pre and post acclimation respectively. During the cold water immersion, TPR at 80 min exposure was 0.011 ± 0.001 and 0.010 ± 0.001 $mmHg \cdot ml^{-1} \cdot min^{-1}$ before and after acclimation respectively.

DISCUSSION

We have previously discussed in a separate report (22) the thermoregulatory adaptations that occurred as a result of the cold acclimation program. Cold acclimation similar to the insulative type exhibited by the Korean breath-hold divers, before the use of wet suits became common practice (21), resulted from the repeated cold water immersion. During the postacclimation cold air exposure the subjects' skin temperatures were significantly lower indicative of insulative cold acclimation. We attributed the lower skin temperature during postacclimation cold exposure to greater cutaneous vasoconstriction mediated by an augmented sympathetic nervous stimulation. A larger increment in circulating norepinephrine during the postacclimation cold exposure was observed and taken as evidence for an

augmented sympathetic nervous activity. We have also reported (27) that insulative cold acclimation, produced by our repeated cold water immersion program, had no measurable effect on body fluid regulation.

The results of the present investigation indicate that insulative cold acclimation produced by repeated, short duration, cold water immersion had relatively minor effects on the respiratory and cardiovascular responses to acute cold stress. Generally, when plotted as a function of time, minute ventilation and cardiac output during cold air or water exposure were not significantly altered by the cold acclimation program. When plotted as functions of various body temperatures, minute ventilation and cardiac output exhibited varying degrees of correlation which were not altered by cold acclimation. However, the blood pressure response to cold water immersion was significantly altered by acclimation.

In agreement with previous studies (1,2,3,4), $\dot{V}_E$ increased during the cold exposure proportionally to the metabolic demand. For exercise of low to moderate intensity (< anaerobic threshold), $\dot{V}_E$ changes as a function of $\dot{V}CO_2$ rather than $\dot{V}O_2$ (30). This close relationship is evident in Table 1 during both the cold air and water exposures. Prior to acclimation, the $\dot{V}_E$-$\dot{V}CO_2$ relationship was steeper ($p<0.05$) during the cold air compared to the cold water exposures. Following the acclimation program, the $\dot{V}_E$-$\dot{V}CO_2$ relationship during cold air exposure was not altered. However, during the postacclimation cold water exposure the relationship was increased. Consequently, the $\dot{V}_E$-$\dot{V}CO_2$ relationship was similar during the postacclimation cold air and water

exposures. Whether the change in the $\dot{V}_E$-$\dot{V}CO_2$ relationship during the cold water immersion represents a physiological adaptation is uncertain. Keatinge and Evans (28) reported diminished ventilatory responses to cold stress following repeated cold water immersions. However, their cold exposures lasted only 20 min and their measurements of $\dot{V}_E$ may have included the hyperventilation produced upon immersion into the cold water. Cooper et al. (5) reported that repeated immersion in cold water (12.0-12.7° C) for five days attenuated the initial ventilatory response. We did not measure $\dot{V}_E$ until after 9 min of cold exposure by which time the initial hyperventilation response was over. Furthermore, our measurements continued until 80 min of cold exposure during which time $\dot{V}_E$ was proportional to $\dot{V}CO_2$. Therefore, following the initial hyperventilatory response, the control of ventilation does not appear to be altered by cold acclimation given that the $\dot{V}_E$-$\dot{V}CO_2$ relationship remained unchanged during cold air exposure pre vs post acclimation.

The pattern of breathing during the cold air and water exposures were similar. The $\dot{V}_E$ increased as a function of both tidal volume and respiratory frequency. Consistent with previous studies (5,29), tidal volume tended to increase proportionally more than frequency. Cold acclimation did not appear to alter the pattern of breathing during cold exposure. As seen in Fig. 3, $\dot{V}_E$ was attained with similar increases of tidal volume (V_T) and frequency before and after acclimation. Hey et al. (30) had reported that the $\dot{V}_E$-V_T relationship was altered when body temperature was increased. They did not examine the effect of body cooling on the $\dot{V}_E$-V_T relationship. Our temperature data (22) indicates that postacclimation in cold air the subjects

were cooler. However, this small decrease in body temperature apparently did not significantly effect the pattern of breathing.

During the cold air exposures prior to acclimation, $\dot{V}_E$ was not related to $\bar{T}_b$ or T_{re} but was moderately correlated inversely to $\bar{T}_{sk}$. In cold air, subjects with the coolest skin had the highest ventilatory response. Postacclimation, in cold air $\dot{V}_E$ was inversely related to $\bar{T}_b$ and $\bar{T}_{sk}$ but not to T_{re}. During cold water immersion pre and post acclimation, $\dot{V}_E$ demonstrated weak to moderate degrees of inverse correlations to $\bar{T}_b$ and T_{re}. The evolution of significant correlations between $\dot{V}_E$ and $\bar{T}_b$ or $\bar{T}_{sk}$ during cold air stress probably results from a greater and more rapid decrease of $\bar{T}_b$ and $\bar{T}_{sk}$ following acclimation. A previous study (4) of these relationships during acute cold stress demonstrated a weak but significant inverse relationship between $\dot{V}_E$ and T_{re}. No relationship was reported between $\dot{V}_E$ and $\bar{T}_{sk}$. The earlier study evaluated these relationships at discrete points of time during the acute cold air (7.7° C) exposure. In the present study, the relationships between $\dot{V}_E$ and various body temperatures was calculated using data collected from 10 to 80 min of exposure. This different method of assimilating the data may account for the different results obtained. Except for the initial ventilatory response to cold exposure (5), it is likely that temperature receptors in the skin and hypothalamus do not uniquely influence the steady-state ventilatory response to cold stress. Rather, these inputs stimulate metabolic and circulatory adjustments in attempts to maintain thermoregulatory *homeostasis*. The ventilatory response is then governed by the metabolic requirements. With the exception of the initial ten minutes of cold air

exposure, acclimation did not alter metabolic responses to cold air or cold water, thus ventilatory responses were not altered.

Upon exposure to the cold, oxygen delivery in response to the increased metabolic demands was provided by significant increases in $(a-\bar{V})\ O_2$ difference and cardiac output. Similar to the ventilatory responses, cardiac output increased proportionally to the metabolic demand. Numerous studies (1,3,8,12,13,17) have reported an increase of cardiac output during acute cold exposure. In cold air and water the increased cardiac output was mainly achieved by larger stroke volumes, since heart rate was slightly elevated only in the cold air. This is consistent with findings of several investigators (3,8,12,13,17). The cold acclimation program did not alter the relationship between cardiac output and metabolic rate nor the stroke volume and heart rate responses. Both before and after acclimation in cold air or water the cardiac output, stroke volume and heart rate responses were not significantly different.

When plotted as a function of various body temperatures, cardiac output demonstrated significant negative correlations only during cold water immersion (Table 2). These correlations suggest that the cooler a subject's body, the greater the cardiac output. Again, instead of suggesting that body temperature per se uniquely governs the cardiac output response to cold stress, the present study along with several prior studies (1,3,8,12,13,17) supports the hypothesis that the cardiac output response is primarily directed by increased metabolism resulting from thermoregulatory adjustments attempting to maintain body

temperature. Since metabolism was essentially unchanged by acclimation, there was no change in cardiac output.

Consistent with previous studies (1,2,8,11,12), prior to acclimation mean arterial blood pressure increased via elevations in both the systolic and diastolic components during the cold water immersions. After acclimation, the mean arterial blood pressure was not systematically altered during the cold water exposure. Budd et al. (10) had observed that after cold acclimation induced by exposure to cold while working in Antarctica, the systolic blood pressure response to cold stress was attenuated, but the diastolic pressure response was not altered. In the present study, during the last cold water immersion mean arterial blood pressure did not increase although cardiac output was increasing and total peripheral resistance was not significantly decreasing over time. We have proposed (22) that postacclimation the subjects maintained a warmer and more highly perfused muscle shell at the expense of a cooler superficial shell. If muscle blood flow resistance decreased while cutaneous blood flow resistance increased then total peripheral resistance may not have changed. The attenuation of the blood pressure response following acclimation may result from the increased perfusion of the muscle shell. Alternatively, the pressor response may have been diminished due to reduced subject apprehension and increased familiarity with the test procedures. Although blood pressure was not measured during the first cold air test, the small rise of mean arterial blood pressure only after 60 min of cold air exposure postacclimation also suggests a diminished pressor response.

Two previous studies (10,15) examined the effect of acclimation on heart rate responses to cold stress. Postacclimation, Budd et al. (10) and Hong et al. (15) both observed enhanced bradycardia during cold air and water exposures respectively. Our results do not demonstrate any significant effect of acclimation on heart rate. The lack of an acclimation effect on heart rate may have been associated with the different acclimation procedures used in these various studies. There was no indication of bradycardia during cold air or water exposure before or after acclimation. Our observations of small increases or no change in heart rate during cold exposure are in agreement with those of Raven et al. (8,12) and O'Hanlon and Horvath (4).

In summary, we evaluated the respiratory and cardiovascular responses to acute cold air and water exposures before and after a cold acclimation program. Consistent with previous reports, ventilation and cardiac output responses were closely related to metabolic requirements arising from thermoregulatory adjustments attempting to maintain body temperature. The pattern of breathing and the manner in which cardiac output was attained was not altered by cold acclimation. Blood pressure responses were attenuated following acclimation during cold water immersion, but total peripheral resistance was not significantly altered. These results suggest that the thermoregulatory adjustments resulting from a cold acclimation program do not alter the control of ventilation or cardiac output, but may alter the control of arterial blood pressure.

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FIGURE LEGENDS

Fig. 1. Mean (±SE) oxygen uptakes, carbon dioxide productions and respiratory exchange ratios as functions of time during cold air exposure prior to and after acclimation program. Values at time 0 min measured during 30 min baseline period, * indicates significant ($p<0.05$) difference pre vs. post acclimation.

Fig. 2. Mean (±SE) minute ventilations, tidal volumes and respiratory frequencies as functions of time during cold air exposure prior to and after acclimation program. Values at time 0 min measured during 30 min baseline period.

Fig. 3. Plot (group mean) of minute ventilation as a function of tidal volume during cold air exposure prior to and after acclimation program.

Fig. 4. Mean (±SE) cardiac outputs, stroke volumes and heart rates as functions of time during cold air exposure prior to and after acclimation program. Values at time 0 min measured during 30 min baseline period.

Fig. 5. Mean (±SE) oxygen uptakes, minute ventilations, and cardiac outputs as functions of time during the first and last cold water immersions. Values at time 0 min measured during 30 min baseline period.

Fig. 6. Mean arterial blood pressures (mean ±SE) as a function of time during the first and last cold water immersions. Values at time 0 min measured during the 30 min baseline period, * indicates significant ($p<0.05$) difference pre vs post acclimation.

TABLE 1. EFFECTS OF COLD EXPOSURE ON CONTROL OF VENTILATION.

Variable	Environment	Pre-acclimation m	b	r	Post-acclimation m	b	r
$\dot{V}_E$-$\dot{V}CO_2$	cold air	39.4	-0.8	0.95**	36.5	0.9	0.92**
	cold water	30.9	2.8	0.97**	35.3	0.7	0.94**
$\dot{V}_E$-$\bar{T}_b$	cold air	2.1	-50.3	0.13	-4.6	178.0	-0.39*
	cold water	-14.2	489.3	-0.63**	-7.5	268.1	-0.42*
$\dot{V}_E$-T_{re}	cold air	0.5	1.2	0.02	-8.1	321.2	-0.28
	cold water	-11.1	428.6	-0.58**	-7.8	308.4	-0.55**
$\dot{V}_E$-$\bar{T}_{sk}$	cold air	-0.9	41.7	-0.50**	-0.9	43.0	-0.75**

* $p<0.05$, ** $p<0.01$, m: slope, b: y-intercept, r: coefficient of correlation.

TABLE 2. EFFECTS OF COLD EXPOSURE ON CONTROL OF CARDIAC OUTPUT.

Variable	Environment	Pre-acclimation m	b	r	Post-acclimation m	b	r
$\dot{Q}$-$\dot{V}O_2$	cold air	6.4	4.3	0.60**	6.0	4.5	0.60**
	cold water	6.7	4.5	0.78**	3.5	6.4	0.45*
$\dot{Q}$-$\bar{T}_b$	cold air	-0.7	33.6	-0.16	-0.9	39.2	-0.26
	cold water	-4.1	143.1	-0.73**	-2.0	73.5	-0.54*
$\dot{Q}$-T_{re}	cold air	0.2	2.5	0.02	-1.9	78.0	-0.24
	cold water	-3.8	148.9	-0.77**	-1.6	68.3	-0.59**
$\dot{Q}$-$\bar{T}_{sk}$	cold air	-0.2	11.8	-0.33	-0.1	10.8	-0.36

* $p<0.05$, ** $p<0.01$, m: slope, b: y-intercept, r: coefficient of correlation.

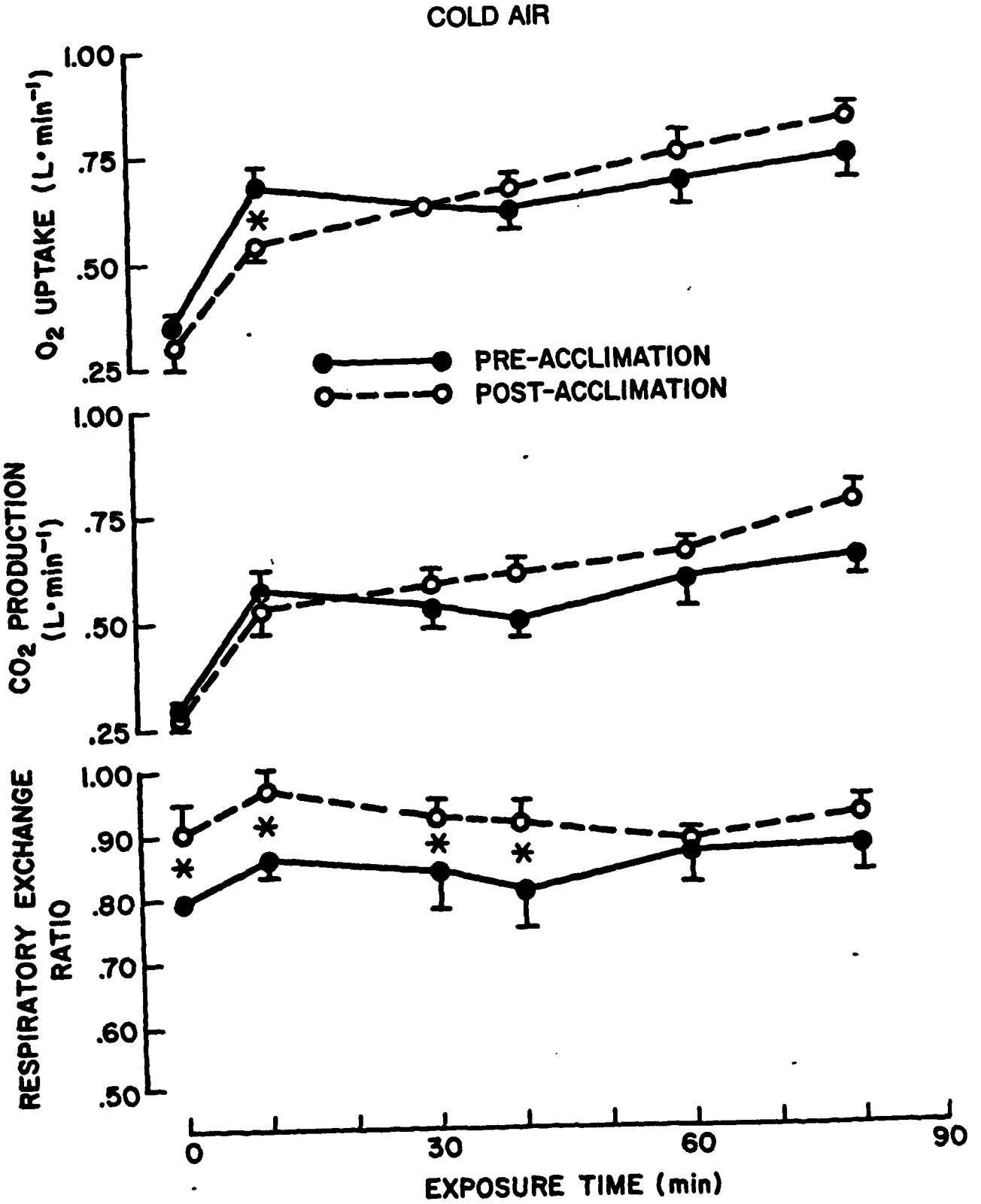

COLD AIR
1.00
.75
.50
.25
O_2 UPTAKE ($L \cdot min^{-1}$)
PRE-ACCLIMATION
POST-ACCLIMATION
1.00
.75
.50
.25
CO_2 PRODUCTION ($L \cdot min^{-1}$)
1.00
.90
.80
.70
.60
.50
RESPIRATORY EXCHANGE RATIO
0
30
60
90
EXPOSURE TIME (min)

Fig 1.

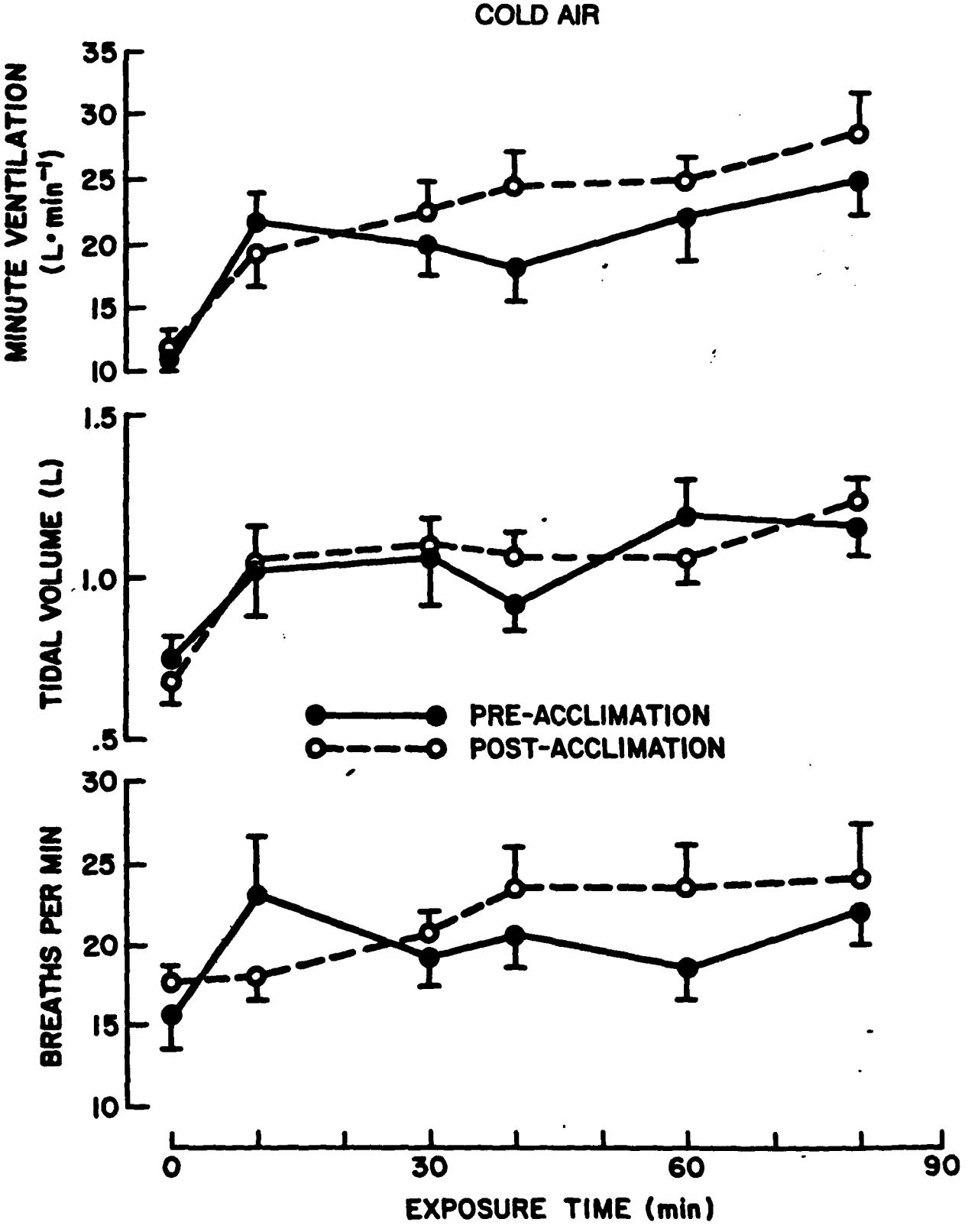

COLD AIR
MINUTE VENTILATION (L·min⁻¹)
35
30
25
20
15
10
TIDAL VOLUME (L)
1.5
1.0
.5
PRE-ACCLIMATION
POST-ACCLIMATION
BREATHS PER MIN
30
25
20
15
10
0
30
60
90
EXPOSURE TIME (min)

Fig 2.

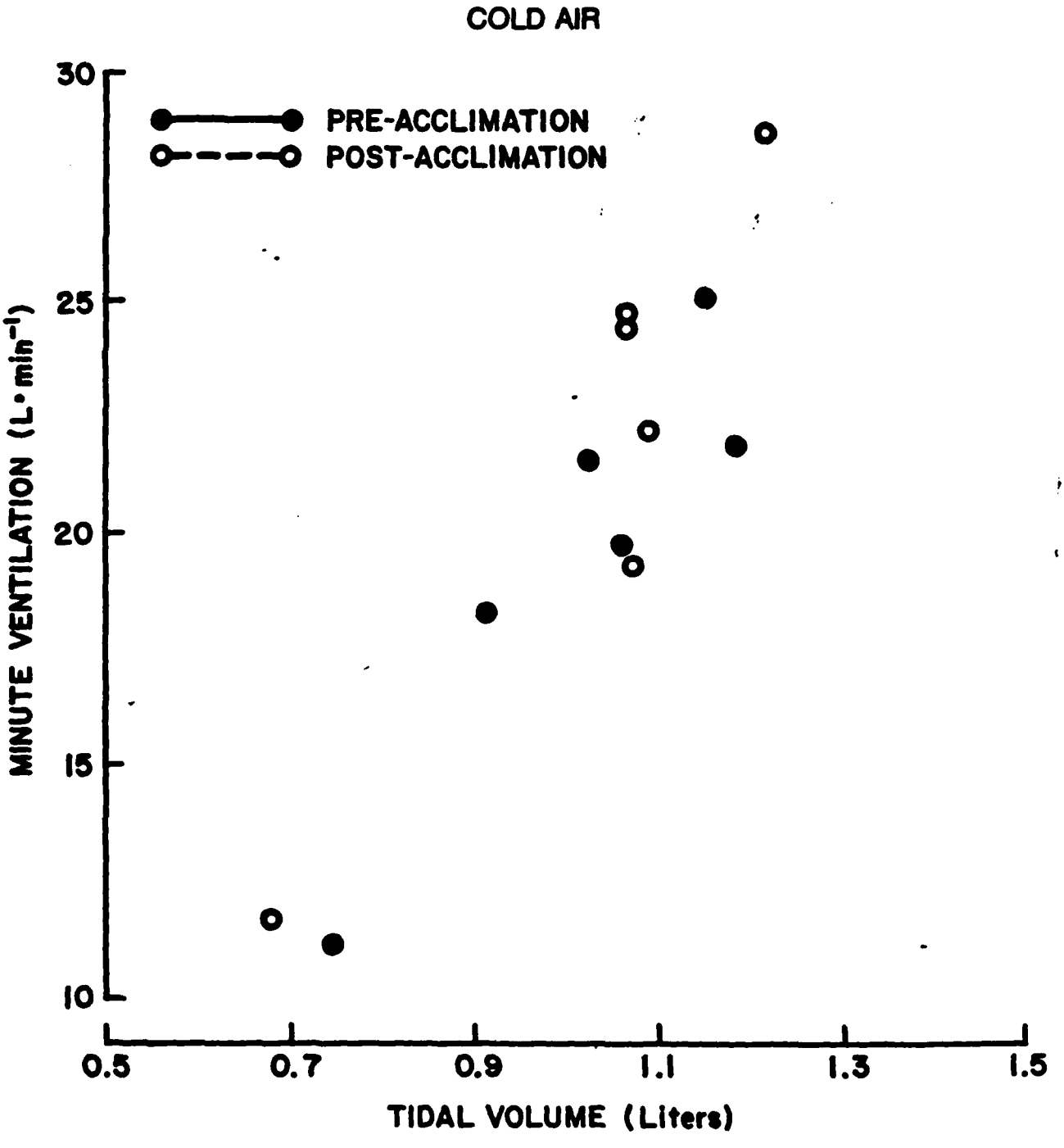

COLD AIR
PRE-ACCLIMATION
POST-ACCLIMATION
MINUTE VENTILATION (L·min⁻¹)
30
25
20
15
10
0.5
0.7
0.9
1.1
1.3
1.5
TIDAL VOLUME (Liters)

Fig 3.

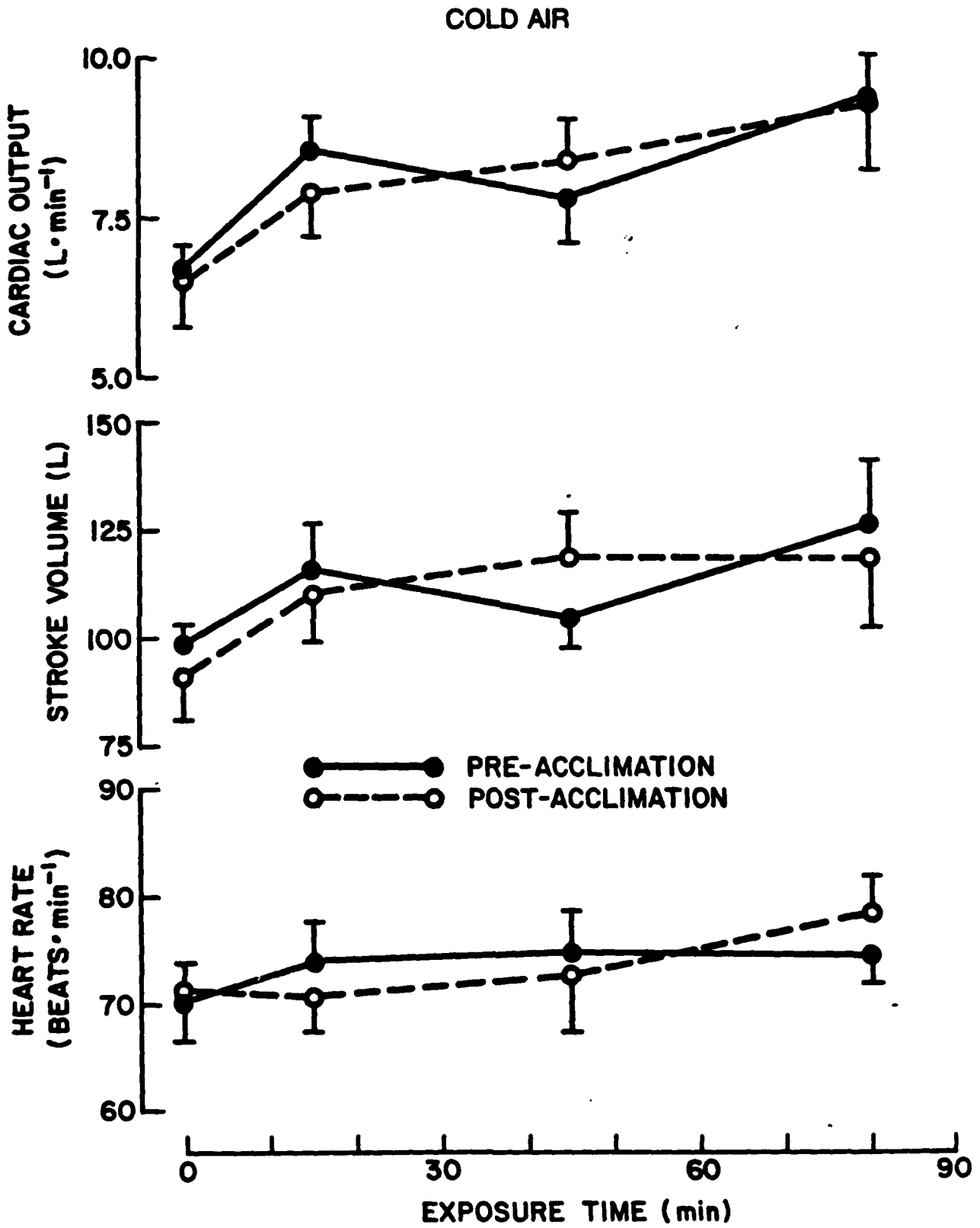

COLD AIR
CARDIAC OUTPUT (L·min⁻¹)
10.0
7.5
5.0
STROKE VOLUME (L)
150
125
100
75
PRE-ACCLIMATION
POST-ACCLIMATION
HEART RATE (BEATS·min⁻¹)
90
80
70
60
0
30
60
90
EXPOSURE TIME (min)

Fig 4.

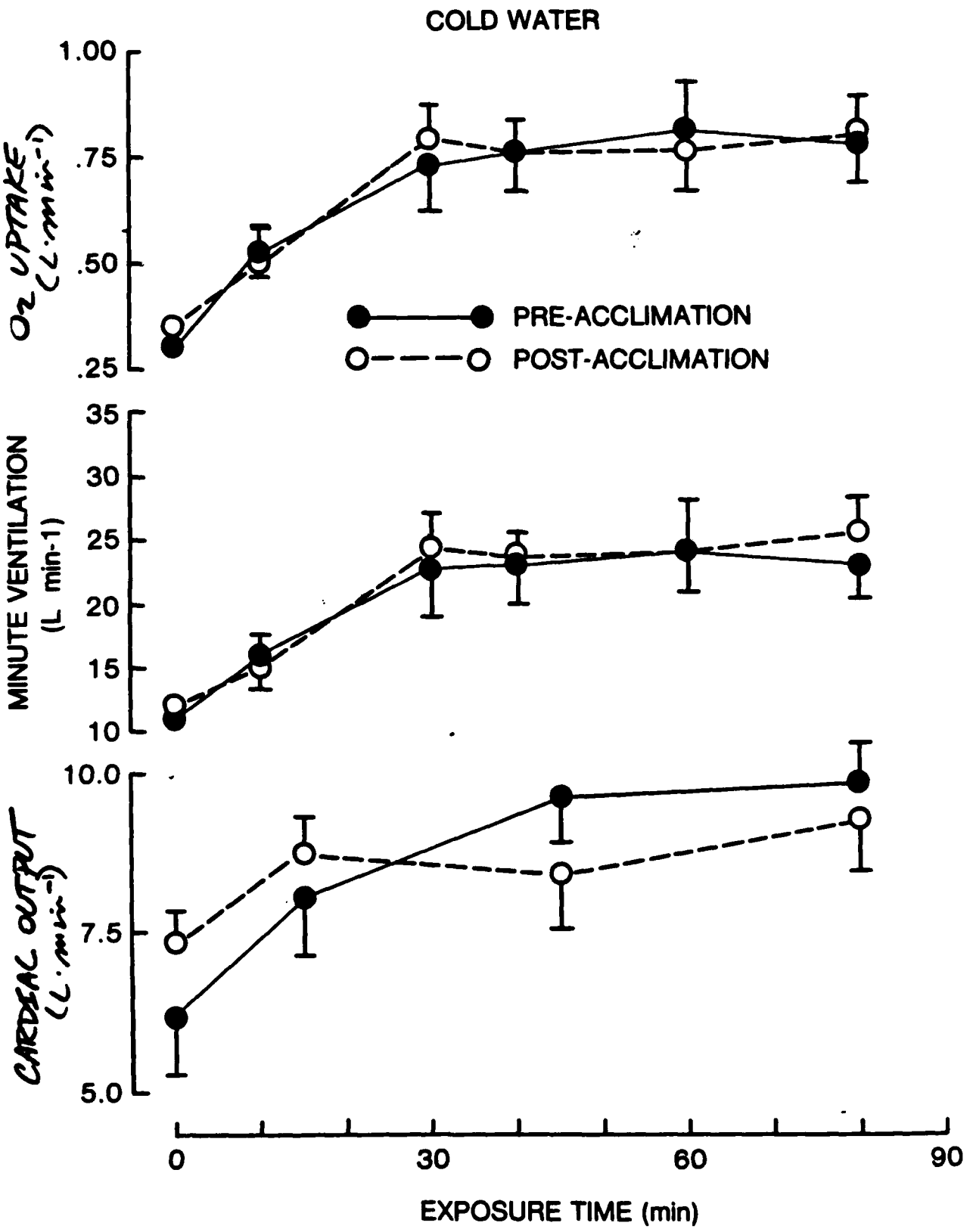
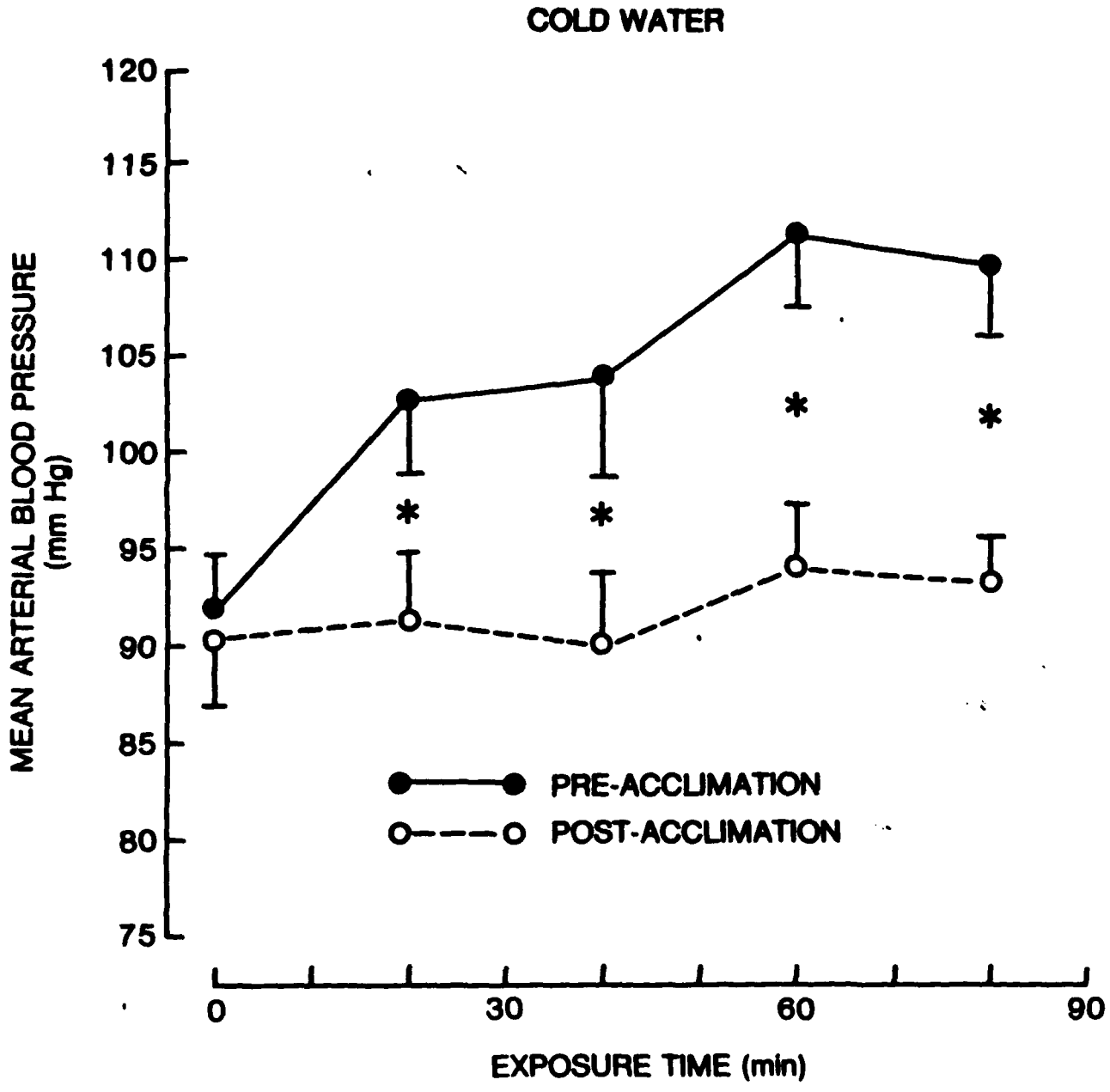

COLD WATER
1.00
.75
.50
.25
O2 UPTAKE (L·min-1)
PRE-ACCLIMATION
POST-ACCLIMATION
35
30
25
20
15
10
MINUTE VENTILATION (L min-1)
10.0
7.5
5.0
CARDIAC OUTPUT (L·min-1)
0
30
60
90
EXPOSURE TIME (min)

Fig 5.

Fig 6.


COLD WATER
MEAN ARTERIAL BLOOD PRESSURE (mm Hg)
120
115
110
105
100
95
90
85
80
75
*
*
*
*
PRE-ACCLIMATION
POST-ACCLIMATION
0
30
60
90
EXPOSURE TIME (min)

END

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